selected for further study: ARP2, Tau, and a neurofilament subunit (XNIF). hnRNP K knockdown in embryos severely compromised the nuclear export and translation but not expression of all three mRNAs, whereas knockdown of the target mRNAs yielded severe defects in axon outgrowth and neuronal cytoarchitecture. These findings are consistent with the idea that hnRNP K is a central element of a larger posttranscriptional regulatory network that orchestrates the highly dynamic expression of functionally interrelated genes that build and organize the axonal cytoskeleton.

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Program/Abstract # 275
Chromosomal regions associated with developmental mutations in congenic inbred lines of chicken
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The University of California–Davis maintains a number of developmental mutant chicken lines which have been studied for their mode of inheritance and phenotypes since the 1950s. These genetic lines segregate mutations causing craniofacial, limb, skeletal, muscular and integument abnormalities. The phenotypes show homology with human developmental syndromes and thus analysis of the lines provide the unique opportunity for identifying causative sequences or genes important in vertebrate developmental biology. The maximum chromosomal region of interest (M_{AX}RI) for ten different developmental mutant congenic inbred lines was determined by genomic analysis utilizing a 60 K single nucleotide polymorphism (SNP) array. Two embryonic lethal recessive mutations, *coloboma* (*co*) and *diplopodia-1* (*dp-1*), were investigated further. *Coloboma* was localized to a 1.6 Mb M_{AX}RI on the Z-chromosome using the SNP chip; *co* results in craniofacial defects, bilaterial facial coloboma, and absent or greatly reduced extremities due to disruption in cartilage formation. The *dp-1* mutant was linked to chromosome 1 (M_{AX}RI = 0.72 Mb); the phenotype includes extra pre-axial digits, dwarfism, and a mild cleft palate. Fine-mapping strategies further narrowed the M_{AX}RI's by over 700 Kb and included new SNP development and analysis of new progeny showing further recombination. Priority candidate genes will be discussed which were established by comparative genomic analysis, i.e., review of the genes in the regions and comparison to the literature in human and mouse.

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Program/Abstract # 276
Transcriptional regulation of cadherin-7 during development of the neural epithelium
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Cadherin-7 (cad-7) shows a dynamic expression pattern during development of a chicken embryo. Cad-7 is expressed in the migrating neural crest cells (NCCs) while premigratory NCCs express cadherin-6B. In neural epithelium (NE), the dorsal domain of the basal plate (lateral NE) expresses cad-7 until stage 23; this expression gradually extends ventrally towards the floor plate by stage 28. This cad-7 expressing lateral domain is part of progenitor cell domains that give rise to V0 and V1 neurons. Given the role of cad-7 during development, it is essential to understand the regulation of its expression. The goal of this study is to identify the gene regulatory elements required for dynamic expression of cad-7. The promoter was identified as a 450 bp region upstream of exon-1 that drives expression of a reporter gene throughout the NE (examined at stages 17–21). However, cad-7 is not expressed in the dorsal and ventral NE at these stages, suggesting the presence of additional regulatory elements. Using tools to identify regions with conserved transcription factor binding site (TFBS), nine evolutionary conserved regions (ECRs) were identified in the intronic and intergenic regions of cad-7 gene locus. ECR-1, containing a 230 bp core functional sequence, together with the promoter drives expression of the reporter gene in a subset of migrating NCCs and the lateral NE similar to endogenous cad-7 expression. The transcriptional activity of the ECR-1 was also confirmed using chromatin immunoprecipitation for acetylated histones in this region. We are in the process of characterizing the active TFBS in this region required for expression of cad-7.

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Program/Abstract # 277
Novel transcription factor involved in neurogenesis
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A novel zinc finger transcription factor, Dmrt5, exhibits temporally and spatially regulated expressions in the developing anterior neuroepithelium in the mouse and the chicken. *In ovo* electroporation of Dmrt5 in chicken embryos causes a reduction in terminal differentiation, as seen by a reduction in beta-tubulin III positive cells. Furthermore, phospho-histone H3 staining indicates overexpression of Dmrt5, but not GFP, causing a reduction in cells going through mitosis. This is accompanied by prominent induction of Hes1, a negative bHLH transcription factor, which when expressed at high levels can inhibit neurogenesis by repressing proneural gene expression [1]. Similarly, overexpression of Dmrt5 during neural differentiation of mouse embryonic stem cells (mES) leads to an upregulation of Hes1, whilst knock out of Dmrt5 during neural differentiation of mES cells causes a reduction in Hes1 expression. Chromatin immunoprecipitation experiments demonstrate that Dmrt5 can bind to the Hes1 promoter, suggesting a direct interaction between Dmrt5 and Hes1. This data indicates that Dmrt5 may regulate neurogenesis by modulating Hes1 levels within the Dmrt5 expressing neuroepithelium. [1] Shimojo H, Ohtsuka T, Kageyama R: Oscillations in notch signaling regulate maintenance of neural progenitors. Neuron 2008, 58:52-64.

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Program/Abstract # 278
MicroRNA-9 regulates neural progenitor proliferation and differentiation in both pallium and subpallium by targeting Foxg1, Nr2e1, Gsh2 and Meis2
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MicroRNA-9-2 and -3 double mutant mouse telencephalons demonstrate that microRNA-9 is required for neural progenitor proliferation and differentiation in both pallium and subpallium by regulating the expression of multiple transcription factors. In the pallium, microRNA-9 suppresses Foxg1 expression for the production of Cajal–Retzius cells and early-born neurons; this suppression is countered by the AU-rich RNA-binding protein Elavl2 at later stages.